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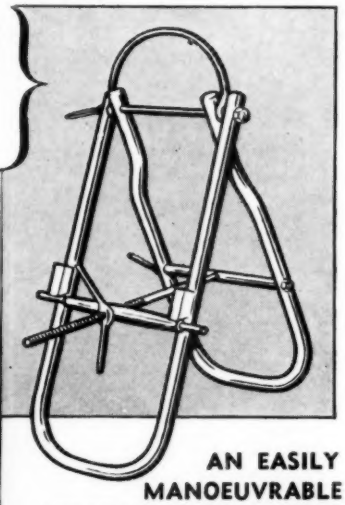

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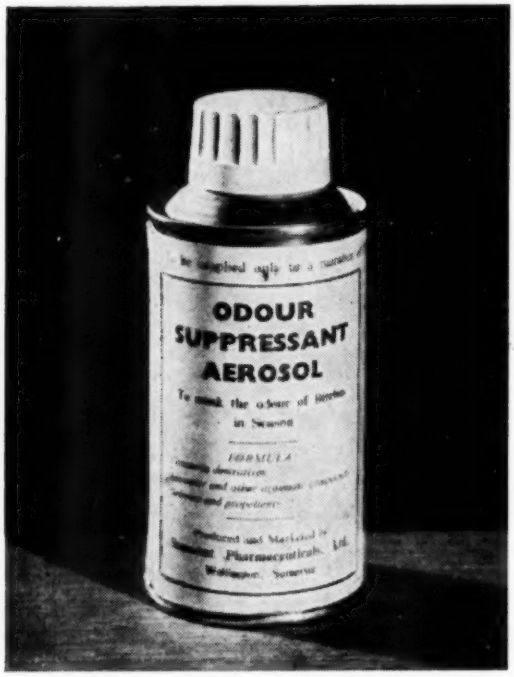

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Museum 6541

A Hitherto Undescribed Disease—Turkey Meningo-encephalitis

BY

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SUMMARY.—A disease affecting turkeys in the Shomron area was investigated. The disease is characterised by progressive paralysis associated with non-purulent meningo-encephalitis. The causative agent of the disease was found to be a filterable virus, cultivable in chicken embryonated eggs. Chickens, ducks, pigeons, hamsters and guinea-pigs were resistant, while turkeys and mice were highly susceptible to the virus. Turkeys which recovered developed immunity to reinfection. Following serial passages of virus in embryonated eggs, a modification in its pathogenicity was noted, both for turkeys and mice. The modified virus retained antigenicity. Preliminary work suggested that an arthropod vector might be involved in the transmission of the virus. Mice immune to turkey virus were found fully susceptible to representative members of group A and B Arbor viruses.

It is suggested that the disease investigated is caused by a hitherto undescribed virus.

IN September, 1958, an outbreak of disease on a large turkey farm was investigated in the Shomron area. Within a short period, clinically identical disease appeared in other turkey farms in the area. Fresh cases continued to appear up to the middle of December when the outbreak apparently subsided. Losses due to the disease are estimated at £25,000.

In September to October, 1959 the disease was again noted in two new widely separated areas, and on one farm affected in the 1958 outbreak. On this last farm survivors of the first outbreak remained unaffected.

The aetiology, clinical manifestations, gross pathology and micropathology of the disease were studied. The causative agent was isolated and characterised. The results of these investigations form the subject of this paper.

Clinical Manifestations

The disease began with the appearance of isolated clinical cases, the number of affected birds in a flock did not reach a peak for 3 to 4 weeks. The spread, if any, of the disease from one affected flock to the next was slow and irregular. In some instances, 6 weeks elapsed before a neighbouring flock was affected, and this was not necessarily one in closest proximity to the first affected flock.

Male and female turkeys of all breeds were found equally susceptible. Young turkeys confined to brooder houses or kept in batteries escaped infection. Exposed chickens and ducks appeared normal.

Affected birds were completely apathetic: they moved with a staggering gait and drooping wings, and produced a greenish diarrhoea. As the disease progressed, complete paralysis of both legs and wings occurred, and the birds lay with their necks extended or twisted in the form of an "S." Progressive drop in egg production was noted in all affected flocks.

Morbidity on the majority of affected farms was above 50 per cent. It was difficult to estimate the mortality associated with the disease as affected flocks were slaughtered at the peak of morbidity. A 10 to 12 per cent. mortality from date of appearance of the disease to date of slaughter was recorded. Recovery occurred amongst birds isolated from the flock.

Gross *post-mortem* lesions were limited to a slight hypertrophy of the spleen and catarrhal enteritis in about 2 per cent. of the birds examined.

The histopathology was characterised by a marked non-purulent lymphocytic meningo-encephalitis.

Studies on the Aetiology of the Disease

Bacteriological examinations of brain and visceral organs of naturally affected birds were negative. Blood smears did not reveal spirochaetes, and brain, liver and spleen smears stained with Giemsa did not reveal Levinthal-Cole-Lillie bodies.

In transmission experiments, the inoculum consisted of a pool of brain and visceral organs from naturally affected birds. Turkeys received 0.1 c.c. 1:10 brain suspension in saline intracerebrally and

1:10 liver and spleen suspension in saline intramuscularly.

Normal turkeys were obtained from farms known to be free of the disease under investigation. Except for some young birds all the turkeys employed were vaccinated on the farm with live virus Newcastle disease vaccine (Haifa strain) and live virus fowl pox vaccine. The age of the turkeys ranged from 5 weeks to 6 months.

Following inoculation, the birds were kept in individual cages in an isolated room. Uninoculated turkeys in the same room served as controls.

Inoculated turkeys were observed daily. Dead birds and those sacrificed *in extremis* were subjected to *post-mortem* examination, and brain, liver and spleen to bacteriological and histopathological examination.

Seven out of 8 inoculated turkeys in the first transmission experiment manifested progressive paralysis. The incubation period following inoculation ranged from 7 to 11 days. In the second transmission experiment involving 5 turkeys 5 to 6 weeks of age, all contracted infection following an incubation period of 4 to 5 days. The longest period of survival after symptoms of disease appeared was 4 days. Gross pathological and bacteriological examinations were negative and histopathological findings were identical with those occurring in natural cases.

Isolation of the Causative Agent in Embryonated Eggs

Attempts at isolating the causative agent in embryonated chicken eggs were made with centrifuged brain and a composite brain, liver and spleen suspension from either naturally or artificially infected birds.

Hatching eggs were obtained from a closed chicken breeding farm and incubated at the Veterinary Institute. Seven-, 10- and 12-day-old embryonated eggs were used in the above order for yolk sac, allantoic and chorio-allantoic membrane routes of inoculation. The dose per egg, irrespective of route, was 0.2 c.c. of 1:10 saline suspension, and the eggs were incubated at 37° C.

In the first serial passages in embryonated eggs, dead embryos, chorio-allantoic membrane, yolk sac and allantoic fluid were harvested and the centrifuged composite suspension in saline inoculated into a second series of eggs by the allantoic route. At higher egg passage level centrifuged suspension of dead embryos only was used and inoculated into the yolk sac. At various egg passage levels (EPL) dead embryos were harvested and a suspension in saline prepared in the usual manner from which 0.1 c.c. of 1:10 dilution of supernatant fluid was inoculated intracerebrally into normal turkeys.

The causative agent of the disease was isolated in embryonated chicken eggs from tissues of naturally affected turkeys in all 3 attempts. On first passage in eggs, embryo mortality occurred between 6 to 10 days and not all embryos died. No difficulty was experienced in isolating the agent from turkeys succumbing to artificial infection and on the first passage in eggs, embryos died between the 2nd and 5th day. As from the second serial passage, practically all inoculated embryos died. The pattern of embryo mortality is given in Table I.

Dead embryos were haemorrhagic, cherry red in colour and the chorio-allantoic membrane of some of the embryos was thickened and contained isolated cloudy spots.

Turkeys inoculated intracerebrally with 0.1 c.c. of 1:10 embryo suspension in saline of the 2nd, 3rd, 5th and 20th EPL showed progressive paralysis on the 4th to 7th day after inoculation.

The microscopic pathology in turkeys inoculated as above was identical with that noted in natural cases. The agent was re-isolated from the above inoculated turkeys in embryonated eggs.

Some Characteristics of the Turkey Disease Agent

The relative efficacy of the chorio-allantoic, allantoic and yolk sac routes of egg inoculation were tested. The inoculum for all routes was the same embryo suspension.

In all tests the yolk sac route appeared to give the most constant results with regard to embryo mortality pattern.

The agent was found distributed in the embryo, chorio-allantoic, yolk sac and allantoic fluid. The EMLD 50 of embryo suspension was $10^{-3.7}$, chorio-allantoic membrane $10^{-3.6}$, yolk sac and yolk $10^{-3.5}$ and allantoic fluid $10^{-2.4}$. Forty-eight serial

TABLE I
SERIAL PASSAGES OF THE CAUSATIVE AGENT IN EMBRYONATED EGGS

Passage number	Route	Inoculum	Embryo mortality—days									
			1	2	3	4	5	6	7	8	9	10
1	A1.	BLS (1)	1/9	—	—	—	—	1/8	—	1/7	1/6	3/3
2	A1.	E, CA, A1. (2)	—	—	—	1/7	1/6	2/4	3/1	—	1/0	—
3	A1.	E	—	—	2/8	2/6	1/5	3/2	—	1/1	—	—
3	YS	E	1/9	3/6	4/2	1/1	1/10	—	—	—	—	—
4	YS	E	—	2/5	4/1	1/0	—	—	—	—	—	—
5	YS	E	1/9	9/0	—	—	—	—	—	—	—	—
6	YS	E	2/8	6/2	1/1	1/0	—	—	—	—	—	—
7	YS	E	1/9	7/2	—	—	—	—	—	—	—	—
8	YS	E	—	6/2	1/1	1/0	—	—	—	—	—	—

BLS = Brain, Liver, Spleen from naturally affected turkeys. E, CA, AL = Embryo, chorio-allantoic membrane, allantoic fluid. 1/9 = 1 died, 9 survived.

TABLE II

Inoculum	Embryo mortality—days										I.C. test in turkeys
	1	2	3	4	5	6	7	8	9	10	
Seitz filtrate embryo suspension EPL 7	—	—	—	—	3/6	1/3	1/4	3/1	—	—	NT
Suspension of embryo dead on fifth day	—	3/7	1/6	(*) 2/4	3/1	1/10	—	—	—	—	Died NPE
Suspension of embryo dead on fourth day	—	—	—	—	—	—	—	—	—	—	Died NPE

* Embryo harvested for passage in eggs or turkeys. NPE = Non-purulent encephalitis. NT = Not tested.

passages of the agent in embryonated chicken eggs by the yolk sac route did not affect the concentration of the agent in the dead embryo suspension. The EMLD 50 remained about the same at low as well as high EPL and ranged between $10^{-3.5}$ to $10^{-4.4}$.

The filterability of the agent through bacteria retaining filters was tested in 3 experiments. Dead embryos were harvested at the 7th, 9th and 24th EPL, 1:10 dead embryo suspension in broth was prepared, centrifuged at 1,500 r.p.m. for 15 minutes and supernatant fluid passed through Seitz E.K. sterilising pads; 0.2 c.c. of the bacteriologically sterile Seitz filtrate was inoculated into a series of embryonated eggs by the yolk sac route. The pattern of embryo mortality was followed and the specificity of the embryo mortality was tested by an additional passage in embryonated eggs and by intracerebral inoculation of dead embryo suspension into normal turkeys.

The bacteriological sterile Seitz filtrate caused specific embryo mortality and the embryo suspension inoculated intracerebrally into normal turkeys, produced the typical disease condition (Table II).

Allantoic fluid and supernatant fluid of 1:5 embryo suspension of EPL 3, 4, 6, 9, 24 and 48 of the turkey disease virus were tested for agglutination ability of chicken and turkey erythrocytes. As a control on the agglutinability of the erythrocytes used in tests, a parallel test was run with same cells using Newcastle disease virus.

Chicken and turkey erythrocytes were agglutinated by Newcastle disease virus, while the turkey disease virus consistently failed to agglutinate erythrocytes.

5,000 units per c.c. of crystalline penicillin G (sodium), 0.1 gr. per c.c. of dihydrostreptomycin (sulphate), 1 mg. per c.c. chlortetracycline* and 0.1 mg. per c.c. oxytetracycline† had no effect on the viability of the turkey disease agent.

Undiluted embryo suspension of various EPL stored in stoppered glass bottles at −18° C. remained viable for at least 63 days.

Host Range

Turkeys

During the course of this work 30 turkeys were inoculated by different routes with dead embryo suspension harvested from low EPL. Of 7 turkeys inoculated both intramuscularly and intracerebrally all sickened, 6 died and 1 recovered; of 10 turkeys inoculated intracerebrally all died; of 13 turkeys inoculated intramuscularly all were affected, 9 died and 4 recovered. Two turkeys inoculated intratracheally remained unaffected. Control turkeys in separate cages or in direct contact with inoculated turkeys remained unaffected.

Chickens, Ducks and Pigeons

Each of 4 pullets, 5 ducks and 2 adult turkeys received 0.2 c.c. of dead embryo suspension (EPL 3) intracerebrally. Only the turkeys sickened and died.

Five 3-week and 11 1-day-old chicks were inoculated intracerebrally with 0.2 c.c. and 6 1-day-old chicks received 0.25 c.c. of the same material intramuscularly. Following inoculation the embryo suspension was titrated in embryonated eggs and the EMLD 50 was $10^{-4.2}$.

Out of the 6 1-day-old chicks inoculated intramuscularly, 2 sickened between the 7th and 11th day after inoculation and virus was re-isolated in embryonated egg. Out of the 11-day-old chicks inoculated intracerebrally, 7 were affected between the 6th day and the 11th day. Virus was re-isolated in embryonated eggs in the only attempt which was made on a chick which sickened on the 7th day and was killed on the 8th day. Histopathological examinations of the brain of the above chick revealed non-purulent meningo-encephalitis.

TABLE III
HOST RANGE

Host	Inoculum	Route	Results
Turkey	EPL 5	I.C.	*2/0
Turkey	EPL 5	L.M.	9/2
Turkey	EPL 5	I.T.	0/2
Turkey	Contact with affected turkey		0/4
Chicken	EPL 3	I.C.	0/4
3 week-old chicks	EPL 13	I.C.	0/5
Day-old chicks	EPL 13	I.C.	7/4
Day-old chicks	EPL 13	I.M.	2/4
Pigeons	EPL 17	I.C.	0/8
Ducks	EPL 3	I.C.	0/4
Suckling mice	EPL 2	I.C.	8/0
Adult mice	EPL 2	I.C.	8/0
Hamster	EPL 3	I.C.	0/8
Guinea-pigs	EPL 3	I.C.	0/8
Suckling guinea-pigs	Mouse passaged virus	I.C.	0/4
Suckling hamster	Mouse passaged virus		

* 2/0 = 2 died, none survived.

*Aureomycin. † Terramycin.

Eight adult pigeons received each 0.2 c.c. dead embryo suspension EPL 17 (EMLD 50 $10^{-3.6}$) and remained unaffected.

Laboratory Animals

Suckling mice were inoculated intraperitoneally with brain suspension from a turkey which succumbed to the inoculated disease (first passage from a natural case). They died or showed paralysis between the 9th and 11th day. Brain harvested from the latter passage was propagated intracerebrally in suckling mice. Virus from the 10th intracerebral passage in baby mice was re-isolated in embryonated chicken eggs and 2 turkeys inoculated intramuscularly with the same material became affected and recovered.

Three-week-old mice were found susceptible following intracerebral inoculation with embryo suspension EPL 2. Adult mice inoculated intracerebrally succumbed between the 6th and 8th day. To date 39 serial intracerebral passages have been carried out in adult mice.

Brain suspension from the 2nd and 3rd intracerebral passage of the virus in adult mice was titrated intracerebrally in 3-week-old mice. The EMLD 50 was 10^{-4} and $10^{-4.4}$, respectively. Histopathological examinations of mice succumbing to the disease revealed non-purulent meningo-encephalitis.

Adult guinea-pigs and hamsters inoculated intracerebrally with embryo suspension EPL 17 (EMLD 50 $10^{-3.8}$) remained unaffected. Suckling guinea-pigs and hamsters were inoculated intracerebrally with mouse-passaged virus and remained unaffected.

Immunity

Turkeys recovered from natural disease and those surviving intramuscular inoculation were immune when challenged simultaneously with normal controls (Table IV).

Attempts were made to detect neutralising antibodies in the sera of immune turkeys which recovered. Serum was mixed with equal amounts of 10-fold dilutions of egg-passaged virus. The serum virus mixture and virus control was kept overnight at +4° C. and then inoculated into series of embryonated eggs using 6 to 8 eggs per dilution.

In all, 6 sera of recovered turkeys were tested. The neutralisation indexes were low. The results for 3 sera could be considered as either negative or, at best, equivocal as the log neutralisation index was above 0.7 and below 1.5. The log neutralisation indexes for the remaining sera under test were 1.5; 1.7; and 2.2, respectively (Table V).

Modification of the Pathogenicity of the Virus

Marked modification in the pathogenicity of the virus in turkeys and mice was noted as from its 24th serial passage in embryonated eggs. Initially the virus lost ability to produce clinical disease following intramuscular inoculation and at the 34th EPL turkeys resisted intracerebral inoculation.

The modification in pathogenicity was not attended by loss of antigenicity and turkeys inoculated either intramuscularly or intracerebrally with the modified

TABLE IV
INTRACEREBRAL CHALLENGE OF RECOVERED TURKEYS

EPL	Route of inoculation and result		Result of I.C. challenge with EPL 6
	I.C.	I.M.	
3	2/0	0/2 / 2/2(x)	0/2
5	8/0	9/2 / 11/11	0/2
20	0/2	0/2 / 2/2	0/2
24	1/0	0/2 / 0/2	0/2
27	—	0/250 / 250/0	0/4
43	0/6 / 6/1	0/8 / 8/0	0/14

2/0 = Two died, none survived.

2/2 = Two inoculated : two affected and two recovered.

TABLE V
NEUTRALISATION TESTS

Virus	Serum	Teat Number / Log NI							
		1	2	3	4	5	6	7	8
Turkey virus	Turkey serum	1·5	NT	1	0·7	1·3	NT	2·2	1·7
	ND serum	0·7	0	0·1	0	0	0	0	0
NDV	ND serum	NT	NT	NT	3·6	NT	7·5	NT	6·5
	Turkey serum	0			0			0	0·5

NT = Not tested.

TABLE VI
PATHOGENICITY OF LOW AND HIGH EGG PASSAGE LEVEL VIRUS IN MICE

EPL	IC inoculation and results	Results of I.C. challenge with EPL 5
11	6/0	—
12	5/1	0/1
20	3/3	0/3
21	6/0	—
31	0/6	0/6
40	0/5	0/5
41	0/5	NT
43	0/30	0/30
48	0/5	0/5
Control	—	6/0

virus resisted intracerebral challenge of the virulent virus.

The modified virus proved to be apathogenic for the mouse As from EPL 31 intracerebrally inoculated mice failed to show symptoms of disease and resisted intracerebral challenge with virulent mouse-passaged virus.

Consideration of a Natural Mode of Transmission

The seasonal appearance of the disease, which coincides with peak insect activity, suggested that an arthropod vector was involved in the transmission of the virus. In order to explore this possibility 2 batches of laboratory raised *Aedes aegypti* mosquitoes were fed on infected embryonated chicken eggs. Ten days later both mosquito batches were ground, treated with antibiotics, and each batch inoculated intracerebrally into mice. One group of mice remained unaffected. Mice of the second group sickened on the 9th day and showed signs of encephalitis. Histopathological examination of the brains of the affected mice revealed a non-purulent encephalitis. The virus was isolated in embryonated eggs from a brain pool of the affected mice.

Identification of the Virus

The possible relationship of the virus isolated from the turkey disease to other avian pathogens was studied.

Clinical, pathological and aetiological studies excluded ornithosis virus. The turkey virus differed from Newcastle disease virus in that it consistently failed to agglutinate erythrocytes. Serum collected from inoculated recovered turkeys not previously vaccinated against Newcastle disease failed to inhibit known Newcastle disease virus in routine haemagglutination inhibition tests. Known Newcastle disease immune serum failed to neutralise turkey virus, and known turkey disease immune serum failed to neutralise Newcastle disease virus.

Preliminary work on transmission suggested that the turkey virus may be a member of the Arbor group of viruses. Attempts were made to identify the virus by titrating intracerebrally representative members of group A and B Arbor viruses in mice susceptible and immune to turkey virus. Special consideration was given to equine encephalomyelitis virus as Woodring (1957) reported natural infection in turkeys with Western E.E. virus in the United States. Virus was not isolated from affected turkeys by Woodring and the diagnosis was based on neutralisation tests. The clinical manifestations of the above disease closely resembled the disease encountered in the Shomron. As the Shomron is an endemic West Nile fever area, West Nile virus was also considered.

The MLD 50 of Eastern equine encephalomyelitis and Western equine encephalomyelitis in turkey virus immune and susceptible mice was found to be about the same (a difference of log 0 to 0 8).

In order to obtain mice immune to Western equine encephalomyelitis the following procedure was used. Adult mice were inoculated intracerebrally with Sindbis virus and remained unaffected. The above mice were then challenged intracerebrally with Western equine encephalomyelitis and, as expected, resisted the challenge dose. The turkey virus was later titrated intracerebrally in the above mice and the mice were found fully susceptible to the virus.

The MLD 50 of West Nile virus and St. Louis virus did not differ significantly in turkey virus immune and susceptible mice.

Acknowledgments.—The authors wish to express their thanks to Drs. Nobel, Neuman and Drill, of the Pathology Department, and Drs. Zamberg, Kahane and Kuperstein, Poultry Disease Officers, for the help rendered in this work.

Reference

WOODRING, F. R. (1957). *J.A.V.M.A.* **130.** 511–513.

POULTRY STOCK IMPROVEMENT PLAN: 1959-60 REGISTER

The Register of accredited poultry breeding stations, accredited hatcheries and standard bred breeders in England and Wales for the accreditation season 1959/60 beginning October 1st, 1959, has now been published. The former Approved Section has been merged into the Accredited Section. The Register is intended to assist poultry keepers to obtain good stock. Except for certain members working in close collaboration with a hatchery, it shows the breeds and approximate number of accredited and standard bred stock on each breeding station.

Copies of the Register may be obtained, post free, on application to the Eggs and Poultry Branch of the Ministry of Agriculture, Fisheries and Food at Whitehall Place, London, S.W.1., or to the county and regional offices of the National Agricultural Advisory Service. Members of the Plan will receive copies direct from county poultry advisory officers. County Lists of accredited breeders, accredited hatcheries and standard bred breeders, are also obtainable from county and regional offices.

Purchasers of accredited or standard bred stock may obtain *post-mortem* examinations free of charge on the carcase of any bird which dies within 28 days of leaving an accredited breeding station or an accredited hatchery. Carcases sent for examination under this arrangement should be accompanied by a statement showing the name and address of the breeding station or hatchery from which the stock was bought and the date on which it was delivered. Carcases must be securely packed in a box with enough absorbent material to prevent leakage of any of the contents during transit; carriage must be pre-paid and the package should bear the name and address of the sender.

Poultry keepers in Cumberland, Westmorland, Northumberland, Durham, Lancashire and Yorkshire should send *post-mortem* specimens to the Veterinary Laboratory, Eskgrove, Lasswade Midlothian (Station: Bonnyrigg, Scottish Region). Specimens from the rest of England and from Wales should be sent to the Central Veterinary Laboratory, New Haw, Weybridge, Surrey (Station: Addlestone, Southern Region).

The Viability of the Extra-uterine (Abdominal) Foetus of Cattle

Case 1.

Non-parturient Torsion and Rupture of the Bovine Uterus

BY

G. H. ARTHUR
Royal Veterinary College, North Mimms

AND

F. H. JENNER
Brockenhurst, Hants.

Subject. A 2-year-old crossbred Guernsey heifer, 8 months pregnant.

History. On November 4th, 1954, the heifer, one of a group of cattle which were seen daily at pasture, looked ill and was not eating. A veterinary examination was requested on November 8th, 1954. This disclosed an unduly large abdomen which was especially prominent posteriorly. It was difficult to make a satisfactory physical examination of the abdomen because of oedema of its postero-ventral wall but it appeared to contain an excess of fluid. An examination of the genital organs, *per rectum,* gave an inconclusive and perplexing result for, whereas an enlarged uterus and fremitus of the middle uterine artery could be palpated, neither foetus nor cotyledons could be found. The following day, November 9th, 1954, the condition of the heifer had deteriorated considerably. It would not get up; its temperature was 101.5° and pulse 120. It had been seen rolling. The abdomen was still large and fluctuating. A rectal examination confirmed a uterine enlargement but again it was impossible to feel the foetus. Additional evidence of pregnancy was afforded by the detection of an intact cervical seal. The cervix seemed to be rather far forward and somewhat displaced laterally. It was not possible to make a diagnosis. The heifer was obviously suffering from severe shock and the opinion was that this was due to internal trauma with probable involvement of the uterus. It was decided to perform an exploratory laparotomy.

Operation. Under field infiltration anaesthesia the abdomen was entered *via* a vertical incision in the left sublumbar fossa. There was considerable intermuscular oedema and the *transversalis abdominis* muscle was extensively ruptured. When the peritoneum was opened about 10 gallons of bloody fluid flowed away. On manual exploration of the viscera, a foetus was found in its membranes, the latter still being attached to the uterus.

After extraction of the calf an examination of the uterus revealed a 360° torsion, and when this was rectified, by turning the organ clockwise, there was found a large transverse tear at the junction of the free and conjoined portions of the left cornu. Part of the afterbirth was removed and the uterus repaired by inserting a continuous Lembert suture of No. 3 catgut. The abdominal incision was closed by means of eversion sutures of 30-lb. nylon. The calf survived for an hour.

Next day the condition of the heifer had worsened and slaughter was advised.

At *post-mortem* examination an extensive latero-ventral abdominal "rupture" and a rent involving nearly half the circumference of the left pregnant uterine horn were confirmed (Fig. 1).

FIG. 1.—Uterus of Guernsey heifer removed at *post mortem*, showing repaired rent at the junction of the free and conjoined portions of the left uterine horn.

Comment. If, as seems probable, the heifer's first obvious illness was associated with the uterine rupture, then the foetus had survived for 5 days in the peritoneal cavity until it was released at operation.

One can only speculate on the chain of events involved in this case. Presumably, as the result of a severe abdominal stress or external trauma the heifer had suffered a ventral rupture. The resulting sudden decrease of abdominal support allowed the uterus to revolve on its longitudinal axis. This torsion would lead to a marked increase of intra-uterine pressure which resulted in uterine rupture and release of the conceptus into the peritoneal cavity. Alternatively, the uterine torsion and rupture may have been due to a sudden convulsive foetal movement in response to an external stimulus.

Case 2.

Preparturient Uterine Rupture

BY

G. H. ARTHUR
Royal Veterinary College, North Mimms

AND

JOHN GRAY
Aylesbury, Bucks.

Subject. A 5-year-old Friesian cow.

History. It had borne 2 normal calves, the last on October 2nd, 1958.

November 9th, 1959. Due to calve; knocked down by some steers. The same evening the owner reported that calving appeared imminent and when, by next morning, there had been no progress, a veterinary examination was made.

November 10th, 1959. Cervix admits only one finger; *per rectum*, 2 legs of a live calf can be felt. The owner was instructed to wait and to report developments. During the next 48 hours, when, unfortunately, the cow was allowed freedom over a large area of pasture and woodland, nothing significant was seen.

November 12th, 1959. Cervix no more dilated; catarrhal uterine discharge; the uterus was partly involuted and contained no calf but there was still marked fremitus of the right uterine artery. Ballottement of the abdomen gave an impression of a solid object which was apparent on both sides. Two explanations came to mind, namely, that the uterus had ruptured, or that the cow had calved unobserved at range. In respect of the latter, a search for the calf was instituted but it ended unsuccessfully. Meanwhile, the cow's temperature was normal but its appetite poor. A 500-mg. chlortetracycline* "oblet" was put into the uterus.

November 13th, 1959. An all-round improvement was obvious with appetite increasing and milk yield rising.

November 17th, 1959. Appetite good, daily milk yield, 3 gallons, but the cow walked with a restricted gait and her temperature was 103.5°. The cervix was now partially open; the uterus was atonic and could not be clearly distinguished but the right uterine artery was definitely smaller than when previously palpated and it had lost its fremitus. Ballottement of the abdomen, which was now resented, disclosed a solid object behind the left costal arch. An intramuscular injection of 3,000,000 units of procaine penicillin was given.

November 18th, 1959. No change in general condition. With 2 men raising the cow's abdomen by means of a pole, it was possible, *per rectum*, to feel a solid object in front of the pelvic brim and it was now decided that a uterine rupture had occurred and that the foetus was free in the abdomen. The cow was transported to the Royal Veterinary College for a laparotomy.

November 19th, 1959. The foetus was now palpable behind the left costal arch so a left-flank laparotomy was decided on.

Operation. The cow was quite bright and kicked sharply when the abdomen was palpated. Twenty minutes before operation she was given a drench of one ounce of chloral. Paravertebral injection of spinal nerves 13T, 1L, and 2L was carried out and a vertical incision made in the middle of the left paralumbar fossa, with the cow standing. On opening the peritoneum, there was not the usual aspiration of air but malodorous, sanguineous, fluid flowed from the wound. The calf was easily identified lying lateral to the ventral sac of the rumen. The amnion was still intact; it was ruptured and the hind legs of the calf brought out of the wound. The dead calf was extracted, followed by the bulk of the foetal membranes. On exploring the abdominal cavity it was found that numerous adhesions had formed between the remainder of the foetal membranes and adjacent viscera, although there was nothing protruding from the uterus. There was widespread serofibrinous peritonitis with copious fluid and large masses of fibrin. Foetal membranous remnants, fibrin, and fluid were removed by a combination of blunt dissection, aspiration, and mopping with sponges. Attention was now directed to the uterus; it showed advanced involution and thus could not be brought to the wound for a complete inspection. There were fibrinous masses adhering to the organ but the rent had healed and its position was not found. The laparotomy wound was closed as one layer by means of vertical mattress sutures of 30-lb. nylon.

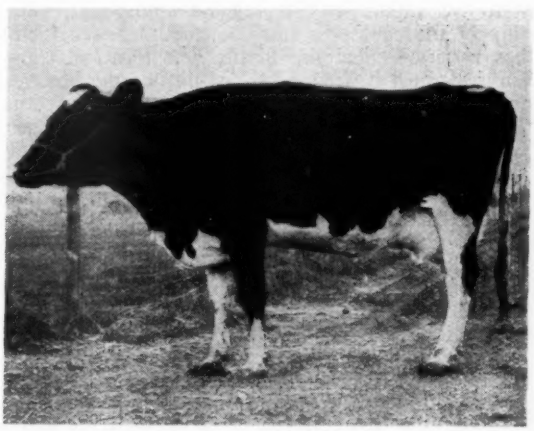

FIG. 2.—Friesian cow, 9 days after left-flank laparotomy, for removal of abdominal foetus.

The cow stood quietly during the operation. It was treated with penicillin for 3 days. For 2 days after the operation it ate little, but from the third day a normal appetite was regained and when the sutures were removed on the ninth day (Fig. 2) the

(*Concluded at foot of col.* 1 *overleaf*)

* Aureomycin.

Clinical Report on the Use of Prednisolone in Sow Agalactia

BY

C. A. G. FELGATE

Chelmsford, Essex

SUMMARY.—The use of prednisolone in agalactia in sows is reported. In febrile conditions this treatment was in combination with antibiotics. In some afebrile conditions the corticosteroid was used without antibiotics.*

The resultant early return to adequate lactation was important not only to the health of the dam, but in preserving the newly born litters which otherwise might have suffered heavy losses due to malnutrition.

* Deltacortril: Pfizer Limited.

LACTATIONAL failure is a frequent post-parturient complication in the sow and may be observed in the presence of infection, with pyrexia and systemic illness, or may be seen as an afebrile condition. The latter form is probably less serious for the sow, but due to the inadequate nutrition of the young pigs and the usual difficulties of fostering newly farrowed litters, either type of agalactia can account for heavy losses in the early days of life.

As a routine treatment a mixed injection of antibiotics with or without pituitrin has been used, but often this has not produced a return to milk which was sufficiently rapid to serve the young pigs.

The following cases illustrate the use of a corticosteroid in conjunction with antibiotics which has given a greatly enhanced response. The addition of 30 mg. of prednisolone to the antibiotic in febrile conditions or the use of the prednisolone alone in afebrile agalactia promoted early response with adequate milk flow, and further treatment was unnecessary.

Case Reports

Case No. 1

The subject in this case was an Essex sow, she had farrowed 2 days prior to the examination on October 25th, 1957, and was found to have one indurated mammary gland and symptoms of anorexia and agalactia. Her temperature was 105° F. A mixed antibiotic injection of dihydrostreptomycin and penicillin together with 30 mg. prednisolone was administered. Next day the sow was revisited, and was found to be eating normally with no pyrexia. The milk flow was adequate. A further dose of dihydrostreptomycin was administered. Uneventful recovery followed, and a healthy litter of pigs was reared.

Case No. 2

The animal in this case was a Blue and White cross-bred sow. When seen on October 25th 1957, she had farrowed early that morning and exhibited a generalised turgidity of the entire udder, with agalactia and normal temperature. Penicillin and 30 mg. prednisolone were injected.

The next day the sow showed some improvement, but it was thought advisable to change the antibiotic to dihydrostreptomycin. Three days later the sow was seen to be completely recovered and suckling 12 piglets.

Case No. 3

On October 27th, 1957, the owner of a Landrace cross Large Black gilt requested examination of this animal which had farrowed 12 hours previously and seemed unable to feed the piglets. The gilt showed

Preparturient Uterine Rupture.—*Concluded.*

milk yield was 21 lb.; this increased later to 38 lb. daily.

Comment. The male calf weighed 73 lb.; it was not obviously putrefying nor emphysematous but some resorption had occurred.

There is little doubt on perusing the history that it escaped from the uterus between November 10th and 12th, that is 7 to 9 days before it was surgically removed. With regard to its period of survival outside the uterus, it seems certain that it died at least a day before atrophy of the uterine artery was noticed on November 17th. This would give a possible maximum survival time of 6 days.

Discussion

The inference from both these cases is that, following rupture of the pregnant uterus and liberation of the conceptus into the abdomen, the foetus has a very limited life in the new location. This survival must depend on the ability of the placenta to satisfy the respiratory requirements of the foetus. Placental function will in turn depend on the integrity of the circulation in, and the physical union between, foetal villi and maternal crypts. Such a union will be adversely affected by uterine contraction; as soon as the foetus escapes, elastic recoil of the uterus diminishes its size by at least one half, and although peristaltic labour contractions may not ensue, involution will soon cause appreciably more shrinkage of the uterine wall and thus jeopardize the cotyledonary circulation.

It is probable also that where uterine rupture occurs near term, chorionic degeneration will, regardless of villus-crypt relationship, itself impose a sharp time-limit on foetal survival. Conversely, a foetus escaping from a uterus ruptured well before term may have a better chance of surviving, and it seems likely that the live calf removed from the Guernsey heifer at five days after uterine rupture was near the limit of its intra-abdominal existence.

In conclusion, it may be said that, in so far as it implies continued foetal development, extra-uterine (abdominal) pregnancy of cattle is a misnomer.

A Case of "Blackquarter" in the Pig

BY

H. A. CLAY

Framlingham

Introduction

SWINE are generally regarded as being relatively resistant to infection with *Clostridium chauvoei* or *Clostridium septicum*. Though cases have been reported in the veterinary literature on a number of occasions, these have usually been encountered on *post-mortem* examination of swine found dead or which have died after only a few hours' observed illness (Orfeur & Hebeler, 1953; Sterne & Edwards, 1955). This case is recorded as evidence of the success resulting from penicillin therapy initiated after early lesions of gangrene were detected.

Clinical Report on the Use of Prednisolone.—*Concluded.*

some turgidity of the udder with agalactia, but had a normal appetite and temperature. 30 mg. prednisolone were injected. Next day the owner reported that the gilt was suckling the piglets quite normally.

Case No. 4

The subject was a Blue and White cross-bred sow, which when examined on November 1st, 1957, was found to have mastitis in several glands, with agalactia, anorexia, and a temperature of 104° F. She had farrowed 3 days previously, and 5 of her 8 piglets were dead. Two grammes dihydrostreptomycin together with 30 mg. prednisolone were injected. The next day the sow was eating well with a normal temperature. Induration of the udder had softened appreciably and she was suckling the 3 remaining piglets satisfactorily; 2 g. dihydrostreptomycin were injected.

Case No. 5

The animal in this case was a cross-bred sow and when seen on November 8th, 1957, had farrowed 8 piglets 2 days before. There was induration of one of the mammary glands with agalactia, and the sow had no appetite and a temperature of 104° F. Two g. dihydrostreptomycin and 30 mg. prednisolone were administered. The following day the milk flow was much improved and the piglets better nourished.

Case No. 6

The subject was a cross-bred sow and on November 8th, 1957, she had a generalised mammitis with agalactia, having farrowed 13 piglets 2 days before. There was no pyrexia, but some degree of anorexia was present: 30 mg. prednisolone and 2 g. dihydrostreptomycin were injected. By the next day the sow's appetite had returned to normal and she had adequate milk flow for the 13 pigs which were all subsequently reared.

Case Report

On August 13th the subject, a Large White gilt, 4 weeks pregnant, had been turned out in the sow paddock in company with 3 other in-pig gilts, to run with about 20 sows. For the first 2 days there had a considerable amount of fighting, and all the freshly added gilts had sustained bites and scratches.

On the morning of August 19th the gilt did not get up when the others were fed, and on being raised she was seen to be lame.

When examined by the writer at 11 a.m. she was recumbent, and was only made to rise with considerable difficulty; she was able to walk but was obviously stiff in the right hind leg. Her temperature was 107° F. and her respirations were accelerated. While examining her for evidence of swine erysipelas a diffuse, irregular area of reddening of the skin over the right ham, about 12 inches in diameter, was observed. In contrast to the rest of the body, this was markedly cold to the touch. The lesion suggested to the writer the appearance of a cow's udder in early gangrenous mastitis. Crystalline penicillin 3 m. units was given subcutaneously.

The gilt was seen again at 6 p.m. when the area of gangrene had extended by about 1 inch all round: the reddening was more marked, and the temperature was 106.5° F. A further injection of penicillin 1.5 m. units was given.

On the following morning the animal appeared more comfortable and had drunk a little milk while lying in her bed. Her temperature was 103.5° F. and her respirations appeared normal. On being forced to stand, lameness was much more severe than on the previous day and no weight was taken on the affected limb. The lesion had again extended slightly, was becoming purple in colour, and showed faint, darker wave markings at its periphery. There had developed a considerable oedema on the inner side of the thigh extending down to the hock. A further injection of penicillin 1.5 m. units was given.

On August 21st the animal was examined again and seemed less bright than on the preceding day. She had refused food and was very reluctant to get up. Lameness was still severe, and the affected skin had faded to a light purple colour and was becoming leathery. A distinct tympanitic sound was noted on percussion, but the crepitation remarked in cattle was not evident, presumably due to the greater thickness of skin overlying the contained gas. A further penicillin injection 1 m. units was given.

On August 22nd the gilt was considerably improved. Her temperature was 102.5° F. She was eating freely, getting up to feed without persuasion, and she walked much more easily. She would take weight on the affected leg when standing. The oedema in the thigh region was decreasing and the skin over the affected part was obviously dead and

going to slough. A final injection of penicillin 1 m. units was given.

Actual separation commenced about a week later and was almost complete by September 3rd (see Fig. 1). A deep slough occurred including all the skin and subcutaneous fat and extending to a varying depth of between ½ and 1 inch into the gluteal muscles.

The patient showed surprisingly little concern at this stage; she walked almost perfectly, behaved otherwise in a completely normal manner, and made a definite gain in bodily condition.

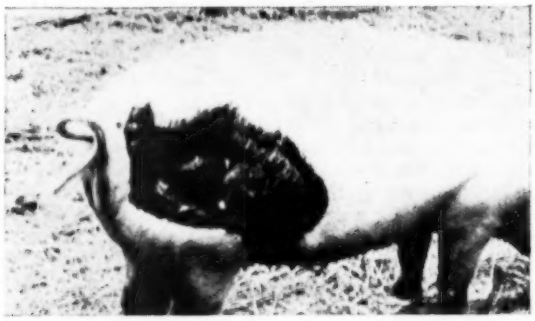

FIG. 1.—"Blackquarter" in the pig. The lesion 2 weeks after onset showing incomplete separation of necrosed skin and muscle.

The open wound was dressed daily by the owner with simple antiseptic lotions and sulphanilimide powder, and healing occurred from the periphery in the course of the next 3 weeks.

Laboratory Investigation

Blood samples were collected on September 14th and October 7th and these were submitted to the Wellcome Research Laboratories for an attempt at identification of the causal organism. Sera from the affected pig and 2 pigs from the same group were assayed by Dr. G. H. Warrack for *Clostridium septicum* α antitoxin, *Clostridium chauvoei* anti-sheep haemolysin and anti-necrotic toxin, and *Clostridium welchii* α antitoxin.

The serum from the affected pig on the first date had a titre of 5 *septicum* α units, <0.01 *welchii* α, and <1 *chauvoei* unit* by haemolytic and necrotic tests. Three weeks later the *septicum* α antitoxin titre had dropped to 1.5 units per ml. The sera from the 2 control pigs had titres of <0.5 *septicum* α, <0.01 *welchii* α, and <1 *chauvoei* unit by haemolytic and necrotic tests.

These findings suggest that the infection was due to *Cl. septicum*.

Discussion

At the time of the gilt's illness only 2 cattle, house cows, were kept on the farm, though, until 3 years before, a dairy herd amounting in all to about 50 head had been maintained for a number of years. As far as is known, no case of blackquarter had occurred previously among cattle or pigs.

In this area the disease in cattle is seen very infrequently and the writer has never previously suspected its occurrence in swine.

Acknowledgments.—I should like to express my gratitude to Dr. G. H. Warrack and Mr. H. B. G. Epps of the Wellcome Research Laboratories for their advice and for carrying out the biological tests.

* Provisional Wellcome "units."

(*Continued at foot of adjoining column*)

References

ORFEUR, N. B., & HEBELER, H. F. (1953). *Vet. Rec.* **65**. 822.

STERNE, M., & EDWARDS, J. B. (1955). *Ibid.* **67**. 314.

Current Literature

BOOK REVIEWS

Deficiency Disease. By R. H. FOLLIS. Blackwell Scientific Publications, Oxford (1958). £5 12s. 6d.

In 1948 Dr. Follis produced a book which, if not the very first of its kind in English, was certainly outstanding; this was his "Pathology of Nutritional Disease." One had looked forward to seeing a second edition of the book, instead of which Dr. Follis has written a new one, with a rather different title and a marked change of emphasis, the sub-title of the new work being "Functional and Structural Changes in Mammalia Which Result from Exogenous or Endogenous Lack of One or More Essential Nutrients." In his preface the author says that whereas his earlier work was concerned largely with the results of *single* nutrient deficiencies and therefore with experimental animals, the present volume also covers multiple nutritional deficiencies such as occur in the field.

There are many ways of assessing the value of a book, but in the present instance there are 3 chief points of significance, namely, what the author has set out to do, how well he seems to have done it, and how useful the product is to the veterinary surgeon.

The first Part of the book consists of some 18 pages on deficiency disease in general, discussing briefly matters such as interference with nutrient intake, absorption or utilisation with increased excretion or requirements, and the problem of nutrient inhibitors.

In Part 2, as in his earlier book, Dr. Follis considers in turn the consequences of deficiencies of about 15 inorganic elements; this takes some 66 pages. Following this is a section devoted to proteins and amino-acids, the consideration of which occupies about 20 pages. Part 4, on lipids, is even more brief, whilst the fifth Part, dealing with carbohydrates, covers 2 pages only. On the other hand the consideration of the vitamins, 17 in all, takes up considerably more space, namely almost 160 pages, and is followed by Part 7, which discusses naturally-occurring deficiency disease, and is about the same length. A considerable portion of this section is devoted to diseases that are chiefly of concern to man, such as kwashiorkor, pernicious anaemia, and beri-beri; even the 20 pages devoted to rickets relate to the human problem.

Part 8 (24 pp.) covers pathological physiology and the anatomy of specific tissues, epithelial, mesenchymal, blood-forming, muscular and nervous, whilst the penultimate section discusses, with extreme brevity, deficiency disease study as a research method in biology and medicine. There follows a useful bibliography of 1,535 references to the literature; and there are also good author and subject indexes.

How useful is this volume to the worker in the field of veterinary medicine? Perhaps some guide is provided by consideration of the numerous Figures, about 170 of which consist of photographs or photomicrographs, mostly of excellent quality. Of that total, 74 concern man himself and 59 the laboratory rat. Trailing behind come the pig (16), the guinea-pig (11), the sheep (3), and the dog, rabbit, and mouse (1 each). One wonders why there should be this kind of bias in a book devoted to "mammalia." It is quite true that it is much easier to establish unambiguous deficiency states in small laboratory rodents than in farm mammals, but since the author's 3 Figures devoted to sheep all concern "swayback," a purely "field disease" until recently, one wonders why the wealth of material relating to other farm animals has been totally rejected. Missing from the index is any reference to parakeratosis in pigs, so that one is not surprised to find no illustration; yet the subject is mentioned (p. 73), though the photomicrographs are all of rat skin. Correspondingly, Fig. 14 illustrates tetany in the magnesium-deficient rat, but one would have thought that the cow would have provided equally good pictures, of interest, to a much wider circle of workers than will ever bother about the rat: it is true, of course, that dietary magnesium deficiency is less well established in the large animal than in the rodent, but surely this matter comes within Dr. Follis's terms of reference, as outlined in his Part 1?

Mention of functional changes in the sub-title of the book aroused the hope that some attention might be given to biochemical aspects of the subject, but the biochemical content of the text seems very small: evidently the adjective refers to general physiological functions.

To the reviewer it appears that Dr. Follis has taken a step closer towards human medicine, as compared with his position when he wrote his earlier work. In consequence his studies of multiple deficiencies seems to have less force for veterinary readers because of the marked contrasts in the relative dietary situations of man and the animals he keeps. To that extent the bibliography is the more important to those who consult this book as a reference work, but, despite the considerable volume of American work in the field of nutrition, one finds with dismay that random sampling of 10 pages of the bibliography produces 185 references to U.S. publications against 53 for the whole of the rest of the world! There are other signs of similar portent. Notable non-American names are missing from the index: reference 1,200 in the text relates to the nineteenth-century recognition of canine blacktongue in Germany, but the reference indicated in the bibliography is a U.S. publication of 1922! At about the same place is a paragraph on tryptophan/nicotinic acid relationships in the pathogenesis of blacktongue: it follows one devoted to the dog, but one has to guess that reference 1,215 is to pioneer British work on the *pig* since the animal species is not indicated in the text. And lastly, the mention of British Units (B.U.) of vitamin A on p. 125 of the text gives the reader a jolt! There is no such unit, although there have been U.S. units: the letters B.U. simply stand for blue units as measured by the Carr-Price test!

This book, then, though useful as a work of reference, within the limits indicated, will be of most interest to the laboratory research worker and to those in the field of human medicine. Though well-produced it is very expensive and, quite frankly, is not the book for which the reviewer had long hoped.

Vitamin B_{12}. By E. LESTER SMITH. Methuen & Co. Ltd., London (1960). 15s.

It is a pleasure to review with unqualified enthusiasm. Much of the enthusiasm in this case stems from the author of this first-class little book.

Dr. Lester Smith, the expert and enthusiast, has performed a remarkable feat in compressing the essentials of a vast and complex literature into so concise yet readable a form. To quote from the General Editor's Foreword, he has produced a book that "can provide an introduction both for the student who is reaching the end of his undergraduate studies and for the research worker who wishes to have an account of a subject cognate with his own."

Vitamin B_{12} is examined and discussed from all angles. The chapter headings are an accurate guide to the contents: Convergent Researches; Origin and Distribution; Isolation; Chemistry; Biogenesis; Nomenclature; Derivatives; Analogues; Antimetabolites; Assay; Binding Factors; and, finally, 4 chapters covering the biological activity of the vitamin and its mechanism of action. Each chapter is subdivided into short sections under clearly defined sub-headings, so that the reader can quickly find information on points of specific interest. The diagrams and tables are straightforward and easily read. Each chapter ends with a list of the significant references, and there is an uncomplicated index at the end of the book.

The style of writing is clear and simple, and the overall impression that remains, as one reaches the last page, is of a job exceedingly well done.

Maladies Infectieuses des Animaux Domestiques. By CH. VAN GOIDSENHOVEN and F. SCHOENAERS. Vigot Frères, Paris, and Sté. Ame. Desoers, Liège (1960). 30 N.F.

It is a matter for regret that so many continental European text-books are produced in a manner not worthy of the care and scholarship they embody. Here is a first-class reference book extending to 852 pages of solid substance (unrelieved, it is true, by any photography), well set out and efficiently indexed; yet it is bound in no more than stiff paper, has a formidable list of errata-addenda, and confusingly bears a title identical with that of Curasson's 2 volumes published in 1946.

XUM

Professors van Goidsenhoven and Schoenaers of the State School of Veterinary Medicine at Cureghem (Brussels), set themselves the task of presenting to students and colleagues an objective picture of our present knowledge in the field of infectious conditions, leaving their readers to make practical deductions from the facts offered them. They have devoted two-thirds of the book to infections of bacterial origin, a further third to viral infections. Each classified infection is dealt with under such headings as Aetiology, Natural and Experimental Susceptibility, Immunity, Pathogenesis, Methods of Infection, Pathology, Bacteriology, Symptomatology, Diagnosis, Prognosis, Prophylaxis, and, where applicable, Treatment. Though 100 pages devoted to bovine tuberculosis may seem outdated by British standards, 26 specific types of abortion in the cow, mare, ewe, goat, and sow are considered in detail, and considerable attention is given to diseases of the newly-born.

This is a most compendious book and a perfect example of the causal school of medicine. The French language is here at its scientific best, each phrase stripped to the bone and tersely evocative. For example, in peracute viral hepatitis of the dog, we read, "A la fièvre intense de début . . . succède l'hypothermie annonciatrice de la mort." Future compulsory reading in the French-speaking veterinary world, this volume could find a place in the library of serious students everywhere.

Report of the Committee of Inquiry on Anthrax. H.M.S.O., London (1959). 12s.

This report is the outcome of the deliberations of a committee appointed in 1957 to consider the existing legal provisions governing the importation of goods likely to be infected with anthrax and the precautions which should be taken to safeguard public health. It is devoted almost entirely to the hazards to workers in industries handling certain imported animal products such as wool and hair, hides and skins, bones and bone meal, and a special chapter deals with workers in docks, wharves and warehouses. The report is a long one and deals with the technical measures taken with certain imported raw materials. The incidence of industrial anthrax is reviewed in great detail and numerous statistical tables are included. Industrial anthrax apparently caused much public concern at the beginning of this century, not so much for its high incidence but because a high proportion of those contracting it used to die. In 1900-1904 21.2 per cent. of human cases ended fatally; but developments in medical treatment in recent years, notably the discovery of antibiotics, transformed the situation, and in 1955-1958 the number of fatal cases dropped to 5.7 per cent. Nearly all cases are of the external type and the incidence of the internal form, which still is almost always fatal, has fortunately declined and is now virtually non-existent. Human anthrax is considered an exclusively occupational disease and the majority of cases occurred in workers handling imported animal products. The remaining cases have all occurred in persons such as farmworkers who could be considered to have caught it while following their occupations. Only one remarkable exception is quoted, that of a clerk in a Pools firm who contracted the disease, and the only possible source of infection which could be suggested was a new woollen jumper worn by the victim shortly before complaining of illness. The committee expressed concern at the risk to agricultural workers, private gardeners, etc., who may handle fertilisers containing raw, unsterilised bone meal. It did not as yet consider that the risk justified compulsory sterilisation, as the cost of £9 10s. or more per ton would make the price prohibitive. The recommendations of the committee include technical measures to be taken with certain imported animal products, aids to speedier diagnosis and treatment, and greater precautions at docks and warehouses and in factories which handle dangerous materials. It also recommended that anthrax should be made a notifiable disease under the Public Health Act. It is notifiable now under the Factories Act and cases among slaughterhouse workers became notifiable to the Chief Inspector of Factories as the result of the passing of the Slaughterhouses Act, 1958. The committee also suggested that some form of immunisation should be considered for workers who may be exposed to infection.

There is little in the report of direct veterinary interest but it throws an interesting light on the complexity of some modern industries. Only passing reference is made to the success of veterinary control among animals in this country and some abroad, such as Australia, New Zealand, and South Africa.

British Medical Bulletin: Antibiotics in Medicine. Volume 16. No. 1. The British Council, London (1960). 20s.

The amount of knowledge that has now accumulated on antibiotics can rightly be described as bewildering. This review succeeds in condensing the important information on this subject into a mere 85 pages—no small achievement. Although written primarily for the medical practitioner, it contains much that is of value to the veterinary surgeon.

The bulletin consists of 15 chapters, all of which are written by experts in their particular fields. Chapters of great interest for the veterinary surgeon include those on the principles of the therapeutic use of antibiotics, their pharmacology, their preventive use in medicine and surgery, and the clinical problems caused by drug-resistant pathogens. To those whose thoughts extend beyond the practical aspects of the treatment of disease, the chapters on the chemistry and classification of antibiotics, the nature of their selective toxicity, the mechanisms of the development of drug resistance and the methods of searching for new antibiotics make fascinating reading.

It would be a remarkably well-informed veterinary surgeon who failed to profit by reading this bulletin, containing as it does such a wealth of summarised information on a subject of great importance.

Infectious Diseases of Animals, Vols. I and II. Diseases due to Bacteria. A. W. Stableforth and I. A. Galloway. Butterworth's Scientific Publications, London (1959). 160s.

One of the most striking features of the post-war veterinary scene has been the steady expansion of veterinary publications ; frequent new editions of old-established textbooks, new books on many aspects of veterinary science, reviews and new journals have all added their quota to the steady growth of veterinary knowledge. The intrinsic veterinary market for literature will always be small and publishers who enter it cannot hope for the pecuniary rewards that may be theirs in the medical field, and yet clearly there is a demand that needs satisfying. Apart from an increase in the veterinary profession in most civilised countries there is also greater interest in our problems, by those concerned with public health, nutrition, and comparative medicine, as well as growing awareness of the overall contribution that veterinary science is making to the economic well-being of mankind.

The first 2 volumes of Stableforth and Galloway's " Infectious Diseases of Animals " are a notable addition to this favourable expansion. It had been the editors' original intention to publish volumes covering diseases associated with bacteria, fungi, Rickettsiae, viruses and protozoa. However, a number of chapters on the 3 latter subjects were not ready and it was decided to publish the first 2 volumes on " Diseases due to Bacteria," including a chapter on Fungal Diseases (Ainsworth). If these 2 volumes are a foretaste of the quality to come we can only urge the editors to persuade their writers to hasten their contributions to the remaining volumes.

The authors of articles in the present volumes are all distinguished microbiologists who, apart from their wide knowledge of their particular fields, have made personal contributions to their subject, and in consequence all the articles have the stamp of authority. The articles are written on the basis of the known microbiology of the disease, but as the editors state in their introduction " the lists of references are not intended to be rolls of honour and cannot be exhaustive." Nevertheless, almost without exception the articles refer to all the relevant recent contributions which were available at the time they were written. (Inevitably the production of such varied material from a number of authors meant a considerable delay between writing and going to press, and there are few references after 1958.)

In all works dependent on a number of authors a certain unevenness is to be expected, but the level is high and no article fails to provide a balanced and up-to-date account of present knowledge. At the risk of appearing invidious the reviewer was particularly impressed by the articles on Brucellosis (Stableforth) ; Tuberculosis (A. B. Paterson, J. T. Stamp and J. N. Ritchie) ; Salmonellosis (A. Buxton and H. I. Field) and Streptococcal Diseases (Stableforth). The volume of the literature on all these subjects is truly formidable and the articles on them are masterly condensations and distillations of information that few of us have the time or energy to acquire for ourselves. The article on brucellosis summarises a mass of data on immunisation and diagnosis that will provide essential background for those who may be involved in any national scheme for the eradication of this disease. The section on the eradication of tuberculosis by J. N. Ritchie is based, as was to be expected, on the overall personal experience of one who can have few equals in knowledge of the problem on a national level. To those concerned with public health, salmonellosis continues to be a problem of daunting dimensions and the article on it in Volume II is an authoritative statement of its veterinary ramifications.

It was logical that the editors should have chosen the aetiological approach to these microbiological diseases and in most instances such an approach gives coherence, but in a disease like bovine mastitis it becomes necessary to read 3 articles : Streptococcal Diseases (Stableforth) ; Staphylococcal Diseases (Williams Smith) and Corynebacterial Diseases (Lovell) to obtain the overall picture. Although Stableforth tries to achieve a balance by his Table 5 on percentage incidence, the article on staphylococci does not really bring out the preponderant influence of these organisms in today's mastitis problem.

This work, according to the introduction, has been designed " for postgraduate students in the veterinary field, in human medicine, and in public health, but hope it will also be of general usefulness to research workers, clinicians, students and others who require a reference work in the field of animal diseases." The editors have every reason to feel confident that their hopes will be justified, for the publication of the first 2 volumes of " Infectious Diseases of Animals " will be one of the outstanding veterinary events of 1960 and they stand as one of the most distinguished British contributions to veterinary literature.

Despite the price, this is a work that those who can benefit from it, which will include most veterinarians, would be well advised to buy for themselves rather than to rely on library copies.

BOOKS RECEIVED

Neurologic Examination of the Dog (With Clinicopathologic Observations), by John T. McGrath, V.M.D. Henry Kimpton, London (1960). 2nd edition. 52s. 6d.

The Urine of the Dog and Cat (Analysis and Interpretation), by Frank Bloom, D.V.M. Gamma Publications Inc., New York (1960). $3.75.

British Medical Bulletin: Antibiotics in Medicine. Scientific Editor. P. Garrod. The Medical Department, The British Council, London (1960). Volume 16. No. 1. 20s.

An Introduction to Veterinary Pharmacology, by Frank Alexander, PH.D., D.SC., M.R.C.V.S. E. & S. Livingstone Ltd., Edinburgh (1960). 20s.

Clinical Veterinary Surgery, Volume II, Operative Procedure, by A. C. Shuttleworth, M.V.SC., F.R.C.V.S., and R. H. Smythe, M.R.C.V.S. Crosby Lockwood & Son Ltd., London (1960). 42s.

Recent Research in Freezing and Drying. Edited by A. S. Parkes, C.B.E., F.R.S., and Audrey U. Smith,

(*Continued at foot of col. 1 overleaf*)

News and Comment

R.C.V.S. DAY LUNCHEON

The principal guest, who will propose the toast of the College at the Annual Luncheon on Wednesday, May 25th at the Savoy Hotel will be the Minister of Agriculture, Fisheries and Food, The Rt. Hon. John Hare, M.P.

Another principal guest who will respond to the toast of the guests, proposed by Professor Weipers, will be Dr. Kenneth Wells, Veterinary Director General of the Dominion of Canada.

Full particulars regarding the Luncheon may be obtained from the Registrar of The Royal College, 9 Red Lion Square, London, W.C.1., who will also give information regarding the Corporate Service and Commemoration of Benefactors Service to be held in the Queen's Chapel of the Savoy, and the Annual General Meeting. These are both held on the same day as the Luncheon.

SUPPLY OF ANTI-ANTHRAX SERUM

The Ministry of Agriculture, Fisheries and Food states that it has discontinued the production of anti-anthrax serum. In special cases veterinary surgeons have been able to obtain anti-anthrax serum from the Central Veterinary Laboratory at Weybridge on the recommendation of the Divisional Veterinary Officer. In future veterinary surgeons should obtain their supplies from commercial sources.

UNIVERSITY NEWS

Edinburgh

The undermentioned candidates have passed in the subject of Veterinary Obstetrics of the Final Professional Examination (Part II) for the degree of Bachelor of Veterinary Medicine and Surgery:—

Abbott, John E. D.; Campbell, Edward G.; Dawson, Peter S.; Downie, James G.; Doxey, David L.; Eyre, Peter; Fairlie, George; Gardner, Edward A.; Gill, Kenneth A. K.; Goad, Lewis; Harcourt, Ralph A.; Irwin, Verney C. R.; Jackson, Peter C. G.; McIlveen, Samuel W. D.; Macmillan, Elizabeth G. M.; Mair, Malcolm L.; Middleton, Nigel T.; Moar, John A. E.; Moore, Alexander; Oduro, Gottfried A.; Pratt, John H.; Radley, David E.; Reeves, Ronald J. C.; Robson, Richard P.; Rollo, David G.; Schermbrucker, Christopher G.; Searles, Patricia A.; Seckington, Ian M.; Smith, Margaret E.; Spence, Albert B.; Taylor, Stuart M.; Thomas, Gruffyd W.; Tindall, John R.; Vaughan-Birch, Allan; Walton, John R.; and Wilkinson, Ian S.

The undermentioned candidates have passed in the subject of Veterinary Preventive and State Medicine of the Final Professional Examination (Part II) for the B.V.M. & S. degree:—

Dawson, Peter S.; Downie, James G.; Doxey, David L.; Eyre, Peter; Fairlie, George; Gill, Kenneth A. K.; Goad, Lewis; Harcourt, Ralph A.; Harrison, Richard D.; Irwin, Verney C. R.; Jackson, Peter G. G.; Lowe, William A.; McIlveen, Samuel W. D.; Macmillan, Elizabeth G. M.; Mair, Malcolm L.; Middleton, Nigel T.; Moar, John A. E.; Oduro, Gottfried A.; Reeves, Ronald J. C.; Robson, Richard P.; Rollo, David G.; Schermbrucker, Christopher G.; Seckington, Ian M.; Smith, Margaret E.; Spence, Albert B.; Taylor, Stuart M.; Thomas, Gruffyd W.; Tindall, John R.; Vaughan-Birch, Allan; Walton, John R.; and Wilkinson, Ian S.

The undermentioned candidates have passed in the Third Professional Examination (Part II) in the subjects of Veterinary Pathology, Bacteriology, and Parasitology for the B.V.M. & S. degree:—

Anderson, Euan C.; Broome, Brenda M.; Carwardine, Paul C.; Cooper, Michael J.; Davies, Charles S.; Foggie, Janet; Fraser, Thomas W.; Glickman, Murray A.; Haigh, Arthur J. B.; Hoskin, Brian D.; Kirk, James O.; Kirkham, John; Lambert, Donald R.; McNab, Ian; Marsh, Ian A.; Mighell, John S.; Muirhead, Michael R.; Pearson, Clifford; Pearson, Michael V. M.; Reece, Peter; Sansi, Kamoru A. O. O.; Stewart, Colin G.; Sutherland, Stewart D.; and Young, Godfrey F.

The undermentioned candidates have passed in the subject of Veterinary Pathology of the Third Professional Examination (Part II) for the B.V.M. & S. degree:—

Arbuckle, James B. R.; Bulley, Frank R. T.; Couthwaite, John R.; Duncan, Walter D.; Gardner, David W.; Hargreaves, Charles E.; and Jobling, Terence.

INTERNATIONAL MEETING ON VETERINARY EDUCATION

It is understood that some 50 governments and international organisations will be sending representatives to the first international meeting of veterinary education. This has been organised by the Food and Agriculture Organisation of the United Nations on the invitation of Her Majesty's Government, and is to be held in Church House, Westminster from April 25th to 29th, 1960.

The organisation in the United Kingdom has been in the hands of a small committee consisting of the President and the Registrar of the Royal College of Veterinary Surgeons, and Mr. Isaacson of the Ministry of Agriculture, Fisheries and Food, together with Sir Thomas Dalling from FAO headquarters in Rome. Any information regarding the meeting may be obtained from Mr. Winterson at the Royal College.

At the end of the international meeting tours have been arranged through the agency of the British Council to visit the veterinary schools of British universities and certain research establishments.

FOWL PEST: RESTRICTIONS IMPOSED IN HUNTINGDONSHIRE

On March 27th restrictions came into force on the movement and marketing of poultry in Huntingdonshire; these restrictions affect all but 12 parishes in the extreme north of the county.

During the last few days outbreaks of fowl pest

Books Received.—*Concluded.*

D.SC., M.B., B.S. Blackwell Scientific Publications Ltd., Oxford (1960). 63s.

The Avian Embryo (Structural and Functional Development), by Alexis L. Romanoff. The Macmillan Company, New York and London (1960). £10 10s.

Principles for British Agricultural Policy (A Study Sponsored by the Nuffield Foundation). Edited by H. T. Williams. Published for the Nuffield Foundation by Oxford University Press, London (1960). 18s.

have been confirmed in the central part of the county and the restrictions are being imposed in order to reduce the risk of further spread of the disease.

ASIAN TYPE OF FOOT-AND-MOUTH DISEASE VIRUS THREATENS EUROPEAN CATTLE

The presence of an exotic type of foot-and-mouth disease virus has been demonstrated in several Near East countries, especially in Syria, the Lebanon and Israel. The report of a recent meeting of FAO's European Commission for the Control of Foot-and-Mouth Disease, says that the virus (type Asia 1) has recently shown a tendency to spread, and that last autumn it caused a severe outbreak in Israel. It was definitely established on that occasion that animals vaccinated against classical types of the virus were not protected from infection by the Asian type.

The report expresses considerable uneasiness about this development, and emphasises that " the greatest danger to Europe now is the introduction of exotic types of the virus." The report also points out that one of the developments of particular importance to the whole of Europe is the presence of foot-and-mouth disease in some Central and Eastern European countries which have been free from outbreaks for several years.

THE BEWICKE AND SHOT PRIZE

We are advised by the Royal Veterinary College that the Bewicke and Shot Prize for 1959 (value £25) has been awarded to R. N. Smith, PH.D., F.R.C.V.S., for his essay entitled " Radiological observations on the developing limbs of Greyhounds."

WELLCOME TRUST BENEFACTIONS

Grants for research made by the Wellcome Trustees for the six months' period September 1st, 1959 to February 29th, 1960, exceed £400.000.

Major grants include benefactions to King's College, University of London, St. Mary's Hospital, University College, London, the University of Toronto, the Johns Hopkins Hospital, Baltimore, and the University College of the West Indies, Jamaica.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

BLAKE, James Cowper, of Graypit, Cypress Road, Newport, Isle of Wight. Graduated New Edinburgh, May 29th, 1899. Died March 24th, 1960, aged 84 years.

JAMES COWPER BLAKE

Mr. G. S. Muir writes:—

Mr. Blake practised in Newport, Isle of Wight, for very many years, and was a well-known sportsman. He was a keen shot, and a supporter of the I.o.W. Hunt of which he was Master for some years. It was largely owing to his efforts that the Hunt was kept alive during the war.

PERSONAL

Mr. F. M. Kearns, M.C., who has been Regional Controller in the Northern Region of the Ministry of Agriculture, Fisheries and Food since April, 1957, will take up a post at the Ministry's headquarters on May 2nd next. He will be succeeded by Mr. A. T. Brooke.

Marriage

CRAWFORD—COOK.—At Banwell, Weston-super-Mare, on March 22nd, 1960, T. Gibson Crawford, M.R.C.V.S., to Peggy M. A. Cook.

Births

HUGHES.—On March 26th, 1960, to Adrienne, wife of David Alan Hughes, B.V.SC., M.R.C.V.S., " Hazeldean," 22 Meadow Drive, Knutsford, Cheshire, a son, Martin David.

RODGER.—On March 27th, 1960, to Jean, wife of John Rodger, M.R.C.V.S., of Ithaka, Hardthorn Road, Dumfries, a son, Hamish.

COMING EVENTS

April

2nd (Sat.). 14th Annual Dinner of the B.R.X. Club at the Royal Veterinary College, London, N.W.1, 6 p.m.

4th (Mon.). Meeting of the East Yorkshire Clinical Club at the Talbot Hotel, Malton, 8 p.m.

6th (Wed.). Spring Meeting of the North of Scotland Division in the Northern Hotel, Aberdeen, 2.30 p.m.

7th (Thurs.). 12th Meeting of the British Veterinary Poultry Association at the Royal Veterinary College, Camden Town, N.W.1, 11 a.m.

8th (Fri.). Meeting of the Midland Counties Veterinary Association at the Abbey Hotel, Great Malvern, Worcs., 2.30 p.m.

Annual Buffet and Dance of the Yorkshire Veterinary Society at the Parkway Hotel, Otley Road, Leeds, 8 p.m.

9th (Sat.). Symposium of the Forensic Science Society at Guy's Hospital Medical School.

12th (Tues.). All-day Meeting of the Central Veterinary Society at the Royal Veterinary College Field Station, North Mimms, preceded by luncheon at the Brookman's Park Hotel, Hatfield, 12.30 p.m.

13th (Wed.). Meeting of the Sussex Veterinary Society at the Old Ship Hotel, Brighton, 2.30 p.m.

19th (Tues.). Joint Meeting of the Scottish Branch with the Society of Practising Veterinary Surgeons in the Golden Lion Hotel, Stirling, 2 p.m.

20th (Wed.). Meeting of the Southern Counties Veterinary Society at the Royal Hotel, Winchester, 7.30 p.m.

Meeting of the Section of Comparative Medicine at the Royal Society of Medicine, 5 p.m.

30th (Sat.). Meeting of the N.W. Region of the B.S.A.V.A. at the Queen's Hotel, Manchester, 7.30 p.m.

Dinner and Dance of the Central Veterinary Society at the Fellows Restaurant of the Zoological Society, Regent's Park, N.W.1, 7 p.m.

Annual General Meeting of State Veterinary Officers in the auditorium of the Wellcome Research Institute, 183-193 Euston Road, London, N.W.1., 4 p.m. The Annual Dinner will follow at the Criterion Restaurant at 6.30 p.m.

XUM

May

6th (Fri.). Annual Dinner of R.V.C.A. at the Royal Veterinary College, Camden Town, N.W.1, 6.30 p.m.

August 28th to September 3rd. 78th Annual Congress of the British Veterinary Association in Glasgow.

B.V.A. COMMITTEE AND COUNCIL MEETINGS IN EDINBURGH

Tuesday, April 19th
Late Evening. Finance Sub-Committee.
Wednesday, April 20th
10 a.m. Small Animals Committee.
12 noon. Home Appointments Committee.
2.15 p.m. Veterinary State Medicine Committee.
4 p.m. Parliamentary and Public Relations Committee.
Thursday, April 21st
10.30 a.m. Farm Livestock Committee.
2 p.m. General Purposes and Finance Committee.
Friday, April 22nd
10.15 a.m. Council Meeting.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak

Anthrax

Angus. Anniston Home Farm, Arbroath (Mar. 28).
Cumbs. Old Town Farm, High Hesket, Southwaite, Carlisle (Mar. 25).
Dumfries. Green Wrae, Gretna (Mar. 28).
Wigtown. Ardewell, Stranraer (Mar. 23).

Swine Fever

Anglesey. Cae Mawr, Beaumaris, Anglesey (Mar. 23).
Berks. Riseley Gorse Farm, Riseley, Swallowfield; Piggery, East Side, Waltham Road, Shottesbrooke, Maidenhead (Mar. 23).
Cambs. Sewards Farm, Chatteris (Mar. 25); Warbraham Farm, Heath Road, Burwell (Mar. 28).
Ches. Oakley Farm, Bradwall, Sandbach (Mar. 23).
Cornwall. Tregoose, Sithney, Helston (Mar. 28).
Devon. Wrights Farm, Payhembury, Honiton (Mar. 24).
Dorset. Piggeries, Wrackleford Lane, Charminster, Dorchester; Quarr Farm, Buckhorn Weston, Gillingham (Mar. 24).
Essex. Sibbards Farm, Debden Green, Debden, Saffron Walden (Mar. 23).
Flint. Bodyrhyddan, Rhuddlan, Rhyl (Mar. 24).
Hunts. 11, Infield Road, Glatton (Mar. 22); The Hawthorns, Station Road, Warboys (Mar. 28).
Kent. The Bungalow, Picardy Manor Way, Belvedere (Mar. 22); Clockhouse Farm, Challock, Ashford (Mar. 23).
Lincs. Broadgate Road, Sutton St. Edwards, Spalding (Mar. 28).
Middlesex. 2, Stacey Cottages, Stockley Road, Stockley, West Drayton (Mar. 23).
Norfolk. Wash Farm, and The Lodge, Denton, Harleston (Mar. 24).
Northants. Levies Farm, Kings Lane, Little Harrowdon, Wellingborough (Mar. 23).
Notts. Commercial Farm, Sutton Bonington (Mar. 28).
Oxon. Hethe Breed Farm, Hethe, Bicester (Mar. 28).
Salop. Priddbwll Mawr, Llansilin, Oswestry (Mar. 22).
Suffolk. Shepherds Farm, Tunstall, Woodbridge (Mar. 23).
Yorks. 11, Main Street, Preston, Hull (Mar. 22).

Moniliasis in Artificially Reared Pigs

We are asked to mention that Dr. Manners's qualifications should have been included in the Contents section last week as follows: M. J. Manners, PH.D., M.SC., N.D.A.

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Dilatation and Torsion of the Bovine Abomasum

Sir,—I have just had the opportunity of reading Messrs. Neil & Pinsent's article on "Dilatation and Torsion of the Bovine Abomasum." They are to be congratulated on this full and careful study of this rare but intriguing condition of the bovine animal. May I add my views and observations which are based on limited clinical experience?

During the past few years I have dealt with at least 9 cases of torsion of the abomasum, and in 5 cases I have had the opportunity of operating. I based my diagnosis on the signs described in the article, and I was invariably impressed by the severe depression and the state of shock in the patient.

I have attempted correction by opening into the abdomen in almost mid-line. With the animal lying on her back under chloral hydrate anaesthesia or narcosis supplemented with local anaesthetic infiltration, an incision of about 7 to 8 inches in length is made 2 inches to the right of the mid-line, almost half-way between the xiphoid cartilage and the umbilicus, through the skin, muscle layers and peritoneum. From this side the abomasum is easily reached for manipulation. After correction the abomasum is fixed to the peritoneum 2 inches to the right of the incision by two interrupted sutures of No. 3 chromic cat gut. The peritoneum and muscle layers are stitched by a continuous suture using "Supramid extra, extra, strong" (in earlier cases No. 3 chromic cat gut was used), and the skin is closed, with edges everted, with 4 or 5 interrupted sutures using the same material. (The same procedure has been used in this practice for some time to correct displacement of the abomasum and it has met with considerable success.)

The following is a record of the 5 cases in which I attempted surgical interference; all were Ayrshire cows which had calved a few days or weeks earlier.

1. and 5. These were peracute cases. Both were violently ill with a grossly distended abdomen especially on the right side. In each case only a small amount of chloral hydrate had been given when the animal collapsed and died. However, laparotomy was performed in the method described and a torsion in a clockwise direction could be felt without difficulty. It is interesting to note that one of these cases was off her food 4 days previously when an assistant found the abomasum displaced to the left side. Without treatment she returned to food the following day and appeared normal until she suddenly became acutely ill with torsion.

2. This animal had been vaguely ill for about 7 to 10 days. One afternoon I decided to carry out an

exploratory rumenotomy and with my hand in the rumen I was able to feel a dilated abomasum and torsion. The same evening the animal was anaethetised and operated on. An assistant and myself were able to identify a clockwise torsion and by raising the mass of the abomasum with the right hand, were able to correct it without much difficulty. We were both impressed by the greatly thickened oedematous wall of the stomach. Within seconds of correction the animal's breathing weakened alarmingly but slowly recovered. On regaining her feet she developed continuous abdominal pain which increased in severity as the effect of the chloral hydrate dwindled. She died within 5 hours. On *post-mortem* examination the torsion was found to have been corrected, the oedema had decreased and there was no ulceration. I was at a loss to account for the pain exhibited.

3. This cow presented all the features of torsion and had been ill for 10 days. Again a clockwise torsion was fairly easily rectified. For 2 or 3 days the animal was very ill but after that she rapidly improved and made a complete recovery.

4. Very similar to No. 3 but she never was quite so ill before or after the operation.

I should like to make the following points :—

a. At one time I thought all cases of torsion of the abomasum were fulminating with resultant death of the animal within a few hours or days. The less severe cases I have described may have been cases of dilatation resulting in torsion, but the animal's clinical picture did not appear to change and torsion was constantly present at operation. Is it not possible that torsion may exist in varying degree resulting in obstruction to a greater or lesser extent and that this, in turn, may dictate the violence of the clinical signs ?

b. In all cases the torsion was clockwise in a vertical plane.

c. I am unable to express an opinion as to the cause of this condition. I think this condition and the much more common but less serious displacement are closely allied. They occur in the same type of animal under the same circumstances and the physical movement of the organ is not dissimilar. In one the abomasum displaces under the rumen to the left side; in the other it displaces to the right side of the rumen on itself and is thus in torsion.

d. The peracute cases are very ill and I doubt if they can be saved by surgical or other means. I commend the mid-line approach under general anaesthesia or narcosis supplemented with a local anaesthetic in cases where an operational risk is justified.

Yours faithfully,
JAMES MILLAR.

Dunure,
Wigtown.
March 19th, 1960.

The Relationship Between Age of Semen and Fertility

Sir,—With reference to the letter from Messrs. Boyd and Reed (*Vet. Rec.* **72**. 189), may I ask the authors to analyse the data in their Table in the way I suggested, *viz.* according to the percentage non-returns at varying intervals after insemination.

Within the limitation of not having the actual data in front of me, I have deduced from their own figures the following interesting information. The "fertilisation" rate or very early embryonic survival as judged by a non-return to heat by 24 days is 8.7 per cent. lower for "old" semen and 14.6 per cent. lower for "frozen" semen when these are compared with 83.8 per cent. holding where fresh semen was used.

Subsequently, however, from 25 to 48 days whilst the apparent embryonic loss for "fresh" semen is 10.4 per cent. as judged by non-returns, it is 16.7 per cent. for "old" semen and 19.4 per cent. for "frozen" semen (expressed as percentages of the remaining non-returns). Thus there is a distinct tendency for the rate of returns to be more rapid at this stage after insemination with "old" and "frozen" semen. The implication is that whilst the apparent fertilisation rates differ, so the degrees of embryonic loss may also vary. In fact, if one is able to interpret the data accurately in this way, the embryonic loss is 6.3 per cent. greater for "old" semen and 9 per cent. worse for "frozen" semen compared with the results obtained from the use of "fresh" semen. From 48 days to calving the comparative loss is 1.7 per cent. for "old" and 3.2 per cent. for "frozen" semen, that is, at a not unduly different rate for the three groups. One would feel that the hazards to the foetus at this later stage would indeed be less where the pregnancy were more established providing the infection were not superimposed.

I wish, therefore, to reiterate my suggestion that wherever differences between the non-return data for groups of animals are established, particularly where the semen has been subjected to different treatments, one would be unwise to ignore the possibility that the data may show quite clear indications of the interaction between two factors—apparent fertilisation rate and apparent embryonic loss.

Yours faithfully,
D. H. L. MADDEN,
Veterinary Officer.
The Hampshire Cattle Breeders' Society Ltd.,
Artificial Insemination Centre,
Lyndhurst,
Hants.
March 22nd, 1960.

"Solubilising".

Sir,—I was recently in receipt of a trade circular including the above word—it was in inverted commas which admitted a certain self-consciousness of its use. To point out that the verb dissolve in the Oxford Dictionary is used to define the adjective soluble, thus leaving no gap necessary for the coinage of a further word, would only add tinder to the fire lit by previous contributors, including you, Sir, against the driftwood accumulating in an adequate language.

But what I would like to remark upon is the lax use of our language combined with an all too free use of the superlative in the claims made for recently marketed drugs—a far more serious charge I feel. How often is it that we receive neatly written charts

or long inclusive lists showing that the " grand little " new drug in question is active against all known varieties of bacteria, viruses and funguses and is particularly active against foot-rot in sheep?

This increasing habit will not only lead to more and more extravagant claims for the marketing of products but better and better salesmanship for their recognition and less and less ethics in their distribution.

Either we ban the use of anything but one qualifying adjective for each noun used in advertisement or we relinguish our non-advertising constipated professionalism and launch full-blooded into the fray of commercialisation on our own account. At the moment our drugs are more appealing than our services (and our charges reflect this).

Yours faithfully,
P. N. TRIM.

16 The Abbey,
Amesbury,
Wilts.
March 20th, 1960.

Warfarin Poisoning at Derby Stadium

Sir,—I, at any rate, was not perturbed that Messrs. Hodgman, Bowden and Wilkinson were all surprised at my letter regarding events at Derby (*Vet. Rec.* **72**. 235, 236). They knew I had been there, and Mr. Wilkinson knew what my views were, though he did not agree with them. I prevailed upon the Derby Stadium to change its feeding methods, and in a letter which followed a diet chart, and enclosing a sample of brown bread, I stated the prothrombin position to the manager. I ended up by writing " and so I am building your diet up, in the belief that in 3 to 5 weeks your cramp and sudden deaths will be a thing of the past." A copy of my letter was sent, as was of course due to him, to Mr. Wilkinson. I took two chances at the time—(1) that warfarin was not being given maliciously, and (2) that the adequate provision of all known vitamins would arrest the episode forthwith. I was so convinced I was right that I even reported in writing to the secretary of the N.G.R.C., that the affair would be ended in a month if my suggestions were carried out; but I added another note to the effect that the cure was so simple it would not be believed.

I don't suppose many people know that the total wastage in the Derby business was 32, of which 9 died, all attributed to warfarin, which I am reliably informed, came from a total amount of bait laid down of 2 lb. or 100 mg. of pure warfarin. It has never been claimed that the amount was either fed direct to the 80 dogs in the kennels, or that the mice kept carrying it on their feet or depositing it all as faeces and urine over the biscuits fed. If they did, and it all reached the dogs in their evenly mixed soaked food, they would each receive 1.25 mg. (It is important to remember that Greyhound food *is* mixed very evenly, because a broth is poured over it, in a tub or bath, and the mixture is twice daily fed as a homogeneous mass). But it is not claimed by anyone that the whole amount of warfarin was fed in one day. The whole process of baiting with warfarin lasted a year, and assuming the mice kept racing quickly from bait to biscuit all that year, we find that, assuming the average weight of the dogs to be 55 lb., each dog had a final total dosage of 0.05 mg. per kg. or a daily dose of 0.05/365 mg. per kg., or approximately 0.00014 mg. per kg. per day. Now it is not pretended that the lot went into the dogs' food, hence the daily amount would be but a fraction of that. As Messrs. Hodgman and Bowden state, " we will never know the quantity consumed," but it is important to remember that it is not claimed warfarin ever got into the food except after passing through the systems of mice or, less clearly, of rats (whose own deaths are not recorded). There was also the mixing process in the preparation of the food. It is further important to remember that 2 Greyhounds died several weeks (4 and 6) after the biscuit said to have been contaminated had been cleared up and baiting had been discontinued. I appreciate that the last dog to die, " Pointed Palm," was somewhat different on *post-mortem* examination, but there was at least a trace of resemblance. He died in full flight, and his abdomen was full of blood, although clotting more rapidly, apparently, than that of his 8 predecessors.

I dislike death in all its forms, but in a way the death of the unfortunate " Pointed Palm " on the day of my visit to Derby was welcome, for it seemed to me to indicate that the process was still going on—as it would have done to anyone else. The *post-mortem* examination was made by Mr. Wilkinson, and findings were as stated. Experience of heart examinations on apparently healthy, destroyed, racing Greyhounds tells me not to overstress the significance of the odd valve vegetation. The important point was why the vessel walls ruptured. All the other cases had died of multiple haemorrhages. The difference here was an apparently improved prothrombin content. The others had a longer clotting time. I submit that " Pointed Palm's " death was basically from the same cause, and was very valuable to all, for it seemed to indicate that the deaths were arising from blood vessel wall impairments, and attention was being focused on the clotting rate which should have been diverted elsewhere.

The following points arise from the correspondence:—

(1) Since the dogs were receiving approximately 1 lb. of biscuit and 1 lb. of meat (albeit overcooked) for a bodyweight of 55 lb., plus water (soup) and doubtless some salt and milk, there is no reason why they should not look well at a casual glance. The deaths at trials and races prove the value one can put on visual inspection prior to racing. I do not in any way blame the veterinary surgeon. " The dogs looked well "—but it emphasises a point and demands our humility.

(2) Comparisons with humans are tricky. My reference to the diet of soldiers was merely to prove that bulk boiling for 3½ hours or over has been incriminated before as a vitamin C killer. The unfortunate experience of the Japanese prisoner must have been trying beyond belief. His original weight may be put at 175 lb.—approximately 3.2 times the

weight of the Derby dog which got 1 lb. of meat and 1 lb. carbohydrate daily. Did the prisoner then get 3.2 lb. of meat and a similar amount of biscuit meal daily? Otherwise let us forget it as a comparison, although remembering he did take vitamin C—a very wise precaution in a deficient diet, whether abroad or at home, in man or in dog.

I do not know of any experimental work for vitamins C and K deficiency in the Greyhound. I think it might be arranged, however, if my principals can be persuaded to divert their annual research subscription for that purpose. There is no doubt we should all welcome the results of such work, for I feel that (a) the Greyhound has been badly neglected in the research world, and (b) as a dog put to hard work he stands in a class of his own, and maintenance diets are insufficient for him.

There is no disagreement between us as to what warfarin will do to the prothrombin level, and we agree no doubt that the Quick technique in no way diagnoses the cause of variations, though it indicates them. I have no reason whatever to doubt that warfarin kills dogs, hence I willingly accept the case of Mr. Shipley's Greyhound (it was positive). I can't be drawn into an argument about the Labrador, it doesn't really concern me—no positive analysis, no proof. We have used warfarin for years, but our dogs haven't died of it yet.

We have plenty to argue about at Derby! With all these carcases available for *post mortem* and analyses, and dogs dying up to February 10th and 27th, 1959, does it not seem significant that in the case of a dog which was received from Mr. Shipley on March 16th (*i.e.* 17 days later) the analyst apparently had no difficulty in reporting it positive to warfarin yet there is no record of a positive result from Derby? It has not been disclosed yet that a sample of biscuit and mice faeces given by Mr. Wilkinson to Dr. Bentley, Principal Scientific Officer of the Ministry of Agriculture and Food, was found to show no trace of warfarin.

What vitamins were missing? I hardly knew what were there! Messrs. Hodgman and Bowden have been "so bold as to assume that I was considering vitamins C, K and K 1." Would that their boldness had seized them earlier! I was indeed, but I did not consider there was nearly enough of any vitamins in the food which reached the dogs, though I believed that the dominating deficiency was vitamin C.

Mr. Wilkinson asks, and reasonably so, why should he change when the dogs had been fed the same way for 10 years or more, without ill effect? The only sparks of vitamin one could definitely point to were *in the milk*, where vitamins A, B and C would be found. I regret any misunderstanding my previous figure of 3 pints per day may have caused. It should have read 8, and I have no hesitation in saying that I was informed by the trainer, in the presence of Mr. Wilkinson and Mr. Hughes, that they gave from "5½ to 8 pints" for 80 dogs. I gave the larger figure, and unfortunately it was wrongly typed. The type of dogs to which it was given may well have been special race dogs; "race dogs" certainly came into it. Not all the dogs were getting it. Sixteen pints of milk for 80 dogs per day is one fifth of a pint—4 oz. each (2 oz. if the figure was 8 pints). In my opinion, the vitamin content of milk is liable to be inconstant, owing to weather conditions in pastures, etc. But I believe that milk probably holds the answer as to how Derby managed to keep going, and how Derby eventually failed without other adequate vitamin support in the complete diet as fed. In fact, the 2 oz. difference between the figure given to me, and that given to Mr. Wilkinson may explain a lot, the ration of fresh milk being much too low for safety. There may have been a trace of vitamin B in the meat, as fed, but it must have been insignificant owing to the prolonged period of cooking. There could have been a trace in the wholemeal part of the baked biscuit, but it too would be low, as the flour appeared to be largely white.

Lastly, Mr. Wilkinson compares race times before and after the episode. Only young dogs improve in their second year of training. Others as a rule gradually lose pace after their second year. But vitamins do not contribute to the energy of the body (Wright). After all, no further deaths took place from the time the diet was changed, and I am gratified that the feeding method continues.

Now, Sir, I have said a lot, but by no means all I would like. Particularly I gloss over what I believe to count most, the effect of deficiency of vitamin C on blood vessel walls, deep haemorrhages, etc. Vitamin K must be left aside, for what is the use of a clotting agent if 2 large renal arteries burst when a dog is running at 30 miles or more per hour? Accepting normal health, and given adequate vitamin content in a suitable diet, the vessel walls will not burst, and the vitamin K will be there. May my friends forgive me for expressing a different interpretation. My point of view was disregarded, but having given it, I felt bound to speak up, even against that array of talent. I could well have written an article on "Avitaminosis at the Derby Track." I hope I have not studied the Greyhound for a lifetime in vain.

May I assure Mr. Wilkinson that I am always very serious on questions affecting the health of this breed.

Yours faithfully,

J. K. BATEMAN.

10 Brackendale,
Winchmore Hill,
London, N.21.
March 26th, 1960.

Reference

WRIGHT, SAMSON. "Applied Physiology."

Gastric Tympany in the Dog

Sir,—It seems possible that Mr. Birkett may be referring to my report last year of a case of gastric tympany in a dog resulting from a diaphragmatic hernia.

If so, he appears to have missed one of the points of the report in that collapse occurred *in spite of* intubation. Respiratory failure occurred during prior anaesthetisation with thiopentone sodium, and although positive pressure *via* a cuffed endotracheal tube was started within a few seconds, cardiac failure also occurred.

It may be of interest to record a similar recent case.

On examination the dog was showing moderated gastric tympany, but although it was making unsuccessful attempts to vomit, it had not reached the stage of acute respiratory distress. The owner, a medical practitioner, attributed the condition to the ingestion of tinned dog meat 3 hours previously. There was again a history of a road accident 2 years previously, and a chest wound penetrating the skin and requiring sutures about 12 months previously. This latter was of unknown origin.

The owner was warned of the possibility of diaphragmatic hernia, but an attempt was made to release some of the pressure by injections of smooth muscle relaxants and antihistamines.

During the next hour some flatus was passed *per anus* but the general condition remained unchanged and a laparotomy was performed.

The dog was anaesthetised with a small amount of thiopentone sodium, and intubated with a cuffed endotracheal tube. Breathing remaind steady, and a ½ in. rubber tube was passed into the oesophagus. On reaching the cardiac sphincter a marked resistance was felt, but this was overcome by gentle rotation of the tube. Immediately there was an explosive release of gas and the abdomen deflated.

The abdomen was opened by a midline incision and the grossly enlarged stomach was found to be lying as described in the previous case, filling the entire abdominal cavity. There was an old rupture of the diaphragm at the junction of ribs and sternum on the left side, and through this rupture small intestine, omental fat and spleen had all passed into the left side of the chest.

Positive pressure was started, and after returning the abdominal contents to their correct position, the rupture was repaired and the abdominal incision closed. An unsuccessful attempt was made to reinflate the left lung.

The dog improved for 3 days under cover of penethamate hydriodide, but then developed pneumonia which caused marked respiratory distress. The temperature rose to 104.7°. Tetracycline in high dosage was given by injection for 3 days and later by mouth. An improvement was noticed on the day following the first injection and has been maintained since, and now, 17 days after the operation the dog appears to his owner to be back to normal. The prognosis must, obviously, still be very guarded.

It is interesting to speculate whether it is a coincidence that both ruptures occurred at exactly the same spot, or whether, and here perhaps the anatomists can enlighten us, there is some inherent weakness at this position.

Yours faithfully,
J. RHODES.

95 Queens Park Parade,
Kingsthorpe,
Northampton.
March 26th, 1960

A Note on Professors

Sir,—May I add a little to the discussion on collective nouns? Dr. Buxton, I believe, is very close to the ideal when he suggests "murmur" as the most suitable collective for our professors; but this, surely, has a suggestion of violence suppressed, of complaints not quite reaching the surface, of a grumbling, niggling attitude. I would suggest "sussuration" as the collective noun of choice exemplified as it is by the quiet swishing noises from the tops of tall trees, so far from the ground, so near to the clouds.

Yours faithfully,
H. E. CARTER.

36 St. George's Road,
Enfield,
Middlesex.
March 22nd, 1960.

Sir,—Surely a "Pomposity of Professors"? Thus, "Professorial Pomposities"!

Yours faithfully,
K. C. SELLERS.

The Animal Health Trust,
Farm Livestock Research Centre,
Stock,
Essex.
March 28th, 1960.

Practice Experience for French Students

Sir,—The Association of Veterinary Students has been asked by the International Veterinary Students' Union to forward the names of practitioners in Britain who would be willing to take a French veterinary student to see practice during the vacations. Would those practitioners who are interested please write to me at the address below?

Yours faithfully,
P. L. BARDGETT,
Secretary,
Association of Veterinary Students.

Liverpool University Veterinary Society,
38 Bedford Street North,
Liverpool, 7.
March 21st, 1960.

Printer's Pie

Sir,—In a footnote to a letter on page 235 of THE VETERINARY RECORD you say that "in correcting one error our printer had the misfortune to make another." Could you tell me in what journal and on what occasion a distinguished officer of the R.A.V.C. was first referred to as a "battle-scared veteran" and then, in an erratum note, as a "bottle-scarred veterinarian"?

Yours faithfully,
C. W. HUME.

The Universities Federation for Animal Welfare,
7a Lamb's Conduit Passage.
London, W.C.1.
March 22nd, 1960.

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